

Medical or Research Professionals/Clinicians

Topic area: *Basic and translational research*

Specific topic: *8. SLE, Sjögren's and APS - etiology, pathogenesis and animal models*

EULAR13-5717

THE CROSSTALK BETWEEN THE COMPLEMENT SYSTEM AND THE COAGULATION CASCADE IN THE ANTIPHOSPHOLIPID SYNDROME. PRELIMINARY DATA FROM BASIC RESEARCH.

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My abstract has been or will be presented at a scientific meeting during a 12 months period prior to EULAR 2013:

No

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Is the first author of this abstract an undergraduate medical student?: No

Background: The association between antiphospholipid antibodies (aPL) and thrombophilic state in antiphospholipid syndrome (APS) is well recognized^[1], but the underlying pathophysiology remains incompletely elucidated.

Several findings suggest the role of complement system (CS) in the pathogenesis of antiphospholipid syndrome (APS)^[2].

The importance of CS in APS is understandable since complement-derived inflammatory mediators increase vascular permeability, activate platelets and promote release of cytokines from monocytes that favor systemic inflammation and coagulation^[3]. It has been demonstrated in a mouse model of aPL-induced pregnancy loss that complement activation can amplify the fetal injury^[4,5]. CS activation has been also documented in patients with APS^[6], but there are far fewer clinical data.

Objectives: To evaluate the relation between CS and coagulation cascade in APS.

Methods: We used plasma samples from healthy women [H] (pregnant: n=5; non pregnant: n=5) and from patients with APS who were non pregnant (n=6), pregnant (n=10), in catastrophic phase [CAT] (n=4) or in a quiescent phase but with the same autoantibody profile of CAT (triple positivity for lupus anticoagulant, anticardiolipin and anti β 2-glycoprotein I antibodies) [SPECIAL] (n=4). The levels of activated complement components (C5a and C5b-9) and Tissue factor (TF) were analyzed using specific ELISA assays.

Results: During this preliminary study, main differences on C5a plasma levels were observed between H and patients with APS (mean H non pregnant: 4.28; APS non pregnant: 10.52), especially in subjects during CAT and in SPECIAL ones (mean 17.38 and 20.49, respectively). No marked differences were observed between pregnant H and with APS (mean APS: 7.97, H: 7.96). Similar data were obtained from C5b-9 analysis.

Plasma TF values in samples from the same women described before were investigated. Among the two subgroups of H women, we didn't observed significant differences in TF values. Conversely, we found interesting differences between non pregnant H controls and patients with APS (mean H: 88.46; APS: 212.21). High values were also documented in APS patients with CAT despite the anticoagulant treatment (mean 168.51), while lower TF values had been observed in pregnant women with APS and in SPECIAL ones (mean 138.17 and 104.65, respectively). We point out that pregnant women with APS and SPECIAL ones received anticoagulant drug to prevent the thrombi formation.

Conclusions: These preliminary data, together with other already reported, support the hypothesis of an involvement of CS in the modulation of coagulation activity in APS, especially in patients with triple positivity.

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Disclosure of Interest: None Declared